editions of English classics which he has picked up. I hear from him every week."

The quiet room, in the sleepy town, the flickering candles, the musty volumes, the devoted father—it was a picture seen through an atmosphere of the teeth-gritting tenseness which pervaded the whole of England at that time; and with a background of that Hell that was being enacted across the Channel—a picture that will not be forgotten.

And it was not long afterward we learned that the dreaded blow had fallen, and that Revere, too, now slept in "Flanders Fields!"

THE DIAGNOSTIC SIGNIFICANCE OF INSPIRATORY MOVEMENTS OF THE COSTAL MARGINS.

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Six years ago the writer published an article[1] on the functions of the diaphragm and their diagnostic significance. In that article former interpretations of phrenic action were criticized and the relation between the curve of the phrenic plane and movement of the costal margins to which it is attached was discussed; experimental and clinical evidence was also presented, to show that inspiratory widening of the subcostal angle and the outward movements of the costal border are not expressions of phrenic excursion but that they are expressions of intercostal activation. It was also shown in this article that when the curve of the phrenic plane is flattened the diaphragm gains the mastery of the costal margin, and that when this is the case the costal margins move toward the median line during inspiration; and, furthermore, that inspiratory narrowing of the subcostal angle is not an evidence of phrenic excursion but purely an evidence of phrenic activation. The only agent which causes widening of the subcostal angle during inspiration is the intercostal musculature.

Four years later[2] the role of the scaleni, intercostals and diaphragm was discussed and the work of Duchenne and several of his contemporaries was reviewed. In this publication it was shown how Duchenne's results came to differ from the results of his contemporaries in performing the same experiment. Further evidence was adduced to show that the costal border phenomena depend entirely upon the plane of the diaphragm, and that when the plane of the diaphragm is convex on its under surface the costal margins

[1] Arch. Int. Med., 1913, xii, 214–224.
[2] Ibid., xx, 701–715.

move outward during inspiration, just as when the diaphragm is convex on its upper surface.

The results of the writer's former observations indicate that the direction and extent of inspiratory movement of the costal margins may be modified by the curve of the phrenic plane, by synechia between the diaphragm and the thoracic wall and by the nerve supply to the intercostal muscles and the diaphragm. These respiratory phenomena are not presented to the examining physician with a surface announcement of the underlying pathology. All that is visible to the examiner is the extent and direction of movement of the outer and inner parts of each costal margin, the movements of the arcs of the ribs and the protrusion of the epigastrium.

A direct method of discussing the role of the diaphragm is to consider the variety of movements of the costal margin during inspiration, and in this article I propose serially to consider the varying movements of these borders.

Exaggerated Symmetrical Outward Movements of Both Costal Margins. Such movements are seen in paralysis of the entire diaphragm, as from poliomyelitis. I have seen one such instance in a child, an account of which was published in the first article referred to. There was great exaggeration of the spreading of the hypochondria, and all of the costal margin of each side moved in an outward direction to an exaggerated degree. Another instance of complete paralysis of the diaphragm was seen in the terminal stages of spinal-cord disease in a woman, aged forty years, who had primary anemia. The exaggeration of the outward movement was due in both these cases to the loss of resistance to the widening movement of the costal margin, which is supplied by a normally acting diaphragm. Under such circumstances there is no opposition to the normal action of the intercostal muscles. Furthermore, the loss of phrenic action demands increased activity of the intercostals to compensate for the loss of phrenic descent during inspiration.

Two other cases were described in which there was exaggerated symmetrical spreading of the subcostal angle, with an exaggerated spread of the hypochondria. In one of these cases the curious movement was due to paralysis of the scaleni in a child with congenital myatonia. All the intercostals and the diaphragm were normal, but, owing to the want of normal anchorage from the scaleni there was an inspiratory retraction of the upper three ribs and manubrium,[3] and the exaggerated outward movement of the costal margins and the hypochondria in this case was a compensatory measure on account of diminution of volume of the upper lung, which occurred during inspiration. This mechanism caused rebreathing between the upper and lower lung, so that the increased spread of the hypo-

[3] Jour. Am. Med. Assn., 1919, lxxiii, 17–20.

chondria and widening of the subcostal angle constituted a purely compensatory measure, which was demanded by the mechanism of rebreathing.

Another patient had the same phenomenon of rebreathing between the upper and lower lung to such an exaggerated degree that it imposed on several medical men as a case of tracheal stenosis. This patient had normally functioning scalene muscles, but had paralysis of the upper four intercostal muscles of both sides. This vicious cycle of rebreathing was intensified just as the respiratory function was called upon for increased movements by exercise or by laughing and crying.

THE ENTIRE COSTAL MARGINS OF BOTH SIDES MOVE SYMMETRICALLY TOWARD THE MEDIAN LINE. This movement is observed (1) in patients who have paralysis of the intercostal muscles and in all instances of injury to the spinal cord in the cervical or upper dorsal region; and (2) in flattening of the diaphragm, due either to chronic emphysema or acute emphysema from bronchiolar spasm. This phenomenon enables us to estimate the degree of flattening of the phrenic leaf. It is invariably present in general bronchiolar spasm and is of service in differentiating between pseudo-asthma and genuine bronchiolar spasm, which occur in patients with chronic bronchitis and emphysema. Many of these patients have attacks which they interpret as genuine asthmatic attacks, when in reality the whole procedure is born of an asthmaphobia. These patients live in constant fear of an asthmatic attack and are liable to have much respiratory discomfort which is psychic in origin. In fact the only manner in which an accurate differentiation can be made between pseudo-asthma and genuine asthma is to observe the costal margins. If the costal margins do not move toward the median line during inspiration then the observer may be perfectly sure there is no bronchiolar spasm, for with bronchiolar spasm there is always sufficient increase of total volume of the lung to flatten the diaphragm sufficiently to procure its mastery over the costal margins.

In the absence of bronchiolar spasm this sign is of great service in estimating the severity of chronic emphysema. When a patient suffering from chronic emphysema, attended with bronchitis, has sufficient enlargement of his lung to give him cyanosis, with a carbon-dioxide percentage in the alveolar air of 7 per cent. or more, there is invariably a sufficient flattening of the entire diaphragm to cause inspiratory narrowing of the subcostal angle and inspiratory retraction of the entire costal margin of both sides. Percussion fails to give us accurate information on this point, because from percussion we can learn only about the descent of the edges of the lung into the pleural sinus, and the pleural sinuses may be completely filled with emphysematous lung and still the entire body of the diaphragm will not be sufficiently flattened to cause inspiratory retraction of the costal margins.

The significance of respiratory retraction of both costal margins was demonstrated in a patient who on entering the hospital breathed fourteen times a minute and had seven liters of tidal air per minute. The patient was cyanotic. The dyspnea and air hunger were very pronounced. The patient's alveolar air, as estimated by the Haldane method, contained 7.3 per cent. of carbon dioxide. This patient had a chronic bronchitis and emphysema, and at the same time did not seem to have bronchiolar spasm. His real difficulty was apparently due to hyperemia of the mucosa of his bronchial path, although this was not proved at the time by the employment of adrenalin or any other drug to show there was no bronchiolar spasm. After two days the patient no longer suffered from air hunger or dyspnea and the cyanosis had disappeared. The patient's alveolar air contained 5.3 per cent. of carbon dioxide instead of 7.3 per cent., as at the time of entrance, but he was still breathing fourteen times a minute, and breathed seven liters of air just as he did at the time of entrance. The difference, however, between the two periods was that on entrance the patient was suffering from intense air hunger and was employing all his strength to ventilate his lung fourteen times a minute, with a half-liter of tidal air at each respiration. In the latter period he breathed at the same rate, with the same tidal air, in perfect comfort. So when this man was at rest he required a seven-liter minute volume of air to properly ventilate his lungs. Now, with the added work which was thrown upon him with stenosis of his bronchioles he demanded, of course, an increase in oxygen consumption, and should have required, as a consequence, a minute volume of air which was beyond his ability to breathe.

This was not the only difficulty with this man. In the first period he had an emphysema which was much severer than during the second period. This, however, was not demonstrable by percussion because at both periods the lower borders of the lung filled the pleural sinus. At the time of entrance to the hospital, however, both costal margins in their entire extent were drawn toward the median line during inspiration; and in the second period, when he was no longer cyanotic, was not suffering from air hunger, and the carbon-dioxide percentage in the alveolar air had diminished 2 per cent., the costal margins were no longer drawn toward the median line during inspiration, but either remained constant in their relation to the median line or showed slightly lateral movements. The movement of the costal borders was the only physical sign on which one could have diagnosed a higher grade of emphysema during the period of dyspnea and cyanosis.

THE ENTIRE COSTAL MARGIN OF ONE SIDE MOVES FARTHER AND MORE PROMPTLY IN AN OUTWARD DIRECTION THAN THE OTHER BONDEN. In observing the costal margin of course one must also take into consideration the movement of the entire arc of the ribs, but when the arcs of the ribs on the two sides move symmetrically,

but the costal margin moves farther on one side, it indicates there has been some accentuation of the arch of the diaphragm on the affected side. This disparity of movement is seen in acute diseases of the liver. On several occasions the writer has observed an increase in the lateral movement of the costal margin of the right side when the patient had an acute swelling of the liver due to parenchymatous disease. This was apparent when the liver did not extend below the costal margin. However, the increase in the outward movement of the margin of the right side indicated there was a greater increase in the volume of liver than the position of its lower border indicated. As these patients recovered from their acute hepatic swelling the costal margins again resumed a perfectly symmetrical movement.

This phenomenon is also seen in subphrenic abscess. The first patient of this kind the writer observed was a man who had had gallstones, was operated, returned home with what seemed a satisfactory convalescence, and reëntered the hospital two months afterward with fever and air hunger. The entire right thorax was flat on percussion from the clavicle to the costal margin. There was a large subphrenic abscess which had ruptured into the pleural cavity, and the liver was displaced nearly a hand's breadth below the costal margin. In spite of total want of pulmonary ventilation on the right side the right costal margin moved laterally much farther than the costal margin of the left side, where there was no disease of the lung or pleura. This exaggerated outward movement of the costal margin on the affected side suggested an impairment of the diaphragmatic influence on the movement of the costal margin, either because the arch of the diaphragm on that side was greatly accentuated by the subphrenic abscess or because of a myositis of the muscular leaf of the right side.

Since this experience the writer has known of five cases of subphrenic abscess, all of which were attended with dulness on percussion, impaired tactile fremitus and bronchial breathing over the base of the right thorax. In all of these cases the diagnosis of subphrenic abscess was made solely on the fact that the costal margin of the affected side moved farther away from the median line than did the costal margin on the healthy side. However, in the course of the formation of a subphrenic abscess, should the diaphragm be displaced in an upward direction, the pleural sinus obliterated, and synechia between the diaphragm and the thoracic wall take place, then a new point of attachment would be formed, so that the effective part of the diaphragm on the right side, namely, that portion which extends from the central tendon to the point of synechia, would have a flattened plane on its upper surface, and instead of the costal margin moving farther away from the median line during inspiration it would move toward the median line. This actually occurred in one case of subphrenic abscess which recently came to operation on

the surgical side of Lakeside Hospital. The costal margin of the right side moved toward the median line during inspiration. A differentiation of thoracic empyema was made as against subphrenic abscess, but at operation a subphrenic abscess was discovered. So far as could be learned at operation the diaphragm was fixed to the thoracic wall, and it was evidently this fixation which gave the misleading sign.

The only supraphrenic or intrathoracic disease which thus far has been observed to give an increased outward movement of the costal margin of the affected side is massive collapse of the lungs. In massive collapse of the lungs we have an acute shrinkage of a large portion of lung without preliminary disease of the affected lung. It has been frequently observed to occur in the lower lobe of each side; and when the lower lobe or both lobes of one side are involved, diminution in volume of the lung is unaccompanied by synechia between the lung and the chest wall or between the chest wall and the diaphragm. This is a rare occurrence and it was little observed prior to the war. With massive collapse of the lower lobe or the entire lung of one side the diaphragm is greatly increased on its upper surface, and although during inspiration there may be no lessening of the vigor of activation of the diaphragm on the affected side, its influence on the movement of the costal margin will have been lessened, and consequently, with lessening traction of the diaphragm on the costal margin, its outward movement will be accentuated.

This is a very valuable diagnostic point in diagnosis of massive collapse of the base of the lung. With massive collapse of the base there will be marked dulness and high-pitched bronchial breathing, with an increased outward movement of the costal margin of the affected side. The differential diagnosis will, of course, lie between subphrenic abscess and collapse of the lung, but in collapse of the lung one can make out other evidences of diminished volume of the affected lung. Not only will the diaphragm be displaced in an upward direction, but the heart and mediastinum will also be displaced toward the affected side. In cases of chronic fibroid disease of the lungs we usually have synechia between the diaphragm and the chest wall. The lung is diminished in volume and increased in density, and there is fixation of the leaf of the diaphragm to the thoracic wall. Here we have not only an upward displacement, but also an upward fixation of the diaphragm, and for this reason chronic fibroid disease of the lung is accompanied by diminished excursion of the costal margin or the costal margin is moved toward the median line during inspiration. Should fibrosis and contraction of the base of the lung be unaccompanied by obliteration of the pleural sinus or synechia between the diaphragm and the chest wall, then, of course, we would have exactly the same physical signs that occur in massive collapse. Wherever this question of differential diagnosis

occurs we have, of course, the previous history to guide us in the differentiation. So thus far collapse of the lower lobe of the lung is the only condition in which the costal margin acquires an increased outward movement during inspiration. Captain M. A. Blankenhorn, of the A. E. F. in France, had ample opportunity to make this observation, and he assures me that the increased outward movement of the costal margin on the affected side was one of the most striking and helpful diagnostic points in making the diagnosis. The essential physical condition for bringing about this unilateral increased movement of the costal margin is a diminution in volume of the lower lobe of the lung unaccompanied by synechia between the diaphragm and the chest wall.

THE COSTAL MARGIN OF ONE SIDE MOVES TOWARD THE MEDIAN LINE. When the costal margin of one side moves toward the median line during inspiration and the other side moves in an outward direction it means that the diaphragm on the affected side has gained the mastery of the costal margin. This will occur when the diaphragm is sufficiently depressed or when the intercostal muscles of the same side are paralyzed. When the dominance of the diaphragm is due to paresis of the intercostals of one side on account of spinal cord disease, there is, of course, no evidence of intrathoracic disease, and not only the ends of the affected rib move toward the median line during inspiration, but the entire arc of the rib will fail to move or will be retracted during inspiration. Unilateral retraction of a costal margin, that is, an entire unilateral movement toward the median line during inspiration, is not a pathognomonic sign of pneumothorax or pleurisy with effusion. It is merely an evidence of the lessening of the normal curve of the phrenic leaf. It does not matter whether the diaphragm is convex on its upper or on its lower surface. In either case the phrenic control of the costal margin is lost. And what is still more significant, it does not matter whether the phrenic leaf of the affected side moves upward or downward during the inspiratory act; the costal margin will move away from the median line if there is sufficient curve to the diaphragm to lose the mastery of the costal margin. This is seen in large effusions into the pleural cavity and also in pneumothorax when the contained air has a pressure above that of barometric pressure. An instance of right-sided pneumothorax, with positive pressure in the pleural cavity and concavity of the upper surface of the diaphragm, was very plainly visible under the fluoroscope. During inspiration the diaphragm on the right moved upward and on the left moved downward. The costal margins on both sides moved away from the median line, but on the right side there was lessened movement in an outward direction. The liver moved upward during inspiration. When the air-pressure was released by paracentesis so that barometric pressure prevailed in the pleural cavity, there was no excursion of the diaphragm during inspiration, but the costal margin of the right side moved strongly toward the median line.

In three instances of large accumulation of fluid in the pleural cavity the spleen was displaced well below the costal margin, the whole left thorax was flat to percussion and tactile fremitus was absent, the respiratory sounds had a bronchial character and there was marked whispered pectoriloquy. The costal margin of the left side moved in an outward direction, but much less than on the right side. With a large accumulation of pus in the pleural cavity one could not see the plane of the diaphragm as in the case of pneumothorax. However, owing to the fact that the entire pleural cavity showed evidences of effusion, paracentesis was performed and two pints of fluid were removed from the pleural cavity. Then the costal margin of the affected side was seen to move strongly toward the median line during inspiration. The pleural cavity was aspirated as completely as possible and the costal margin again moved in an outward direction, although it did not move as far as the costal margin of the opposite side.

These experiences prove two very important points: One is that the direction in which the costal margin will move does not depend upon the position of the diaphragm but on the curve of its plane. The result is quite the same whether the convexity is on the upper or lower surface. These experiences also prove that the outward movement of the costal margin of the affected side does not depend on the descent of the viscera during inspiration. As was seen in the case of pneumothorax, when the liver moved upward during inspiration the movement of the right costal margin was away from the median line.

All these signs may sometimes fail us in making a diagnosis. For instance a woman recently under my observation had a subphrenic abscess of the left side, with fetid empyema, attended with the evolution of gas in the pleural cavity. The patient had all the signs of pyopneumothorax and the costal margin of the affected side moved outward during inspiration. An operative procedure was undertaken; the eighth rib was resected in the axillary line, fetid pus in large amounts was evacuated and a marked convexity of the diaphragm could be clearly seen. Paracentesis through the diaphragm revealed a large subphrenic abscess. Now, in this instance we had a combination of subphrenic abscess with pyopneumothorax of the same side. The position of the diaphragm, so far as we could determine, was pretty nearly that of the normal arch; so the loss of movement of the costal margin on the affected side was due entirely to the want of ventilation of the lungs and the consequently impaired movement of the ribs in their entire extent, but what movement of the costal margin was apparent was in an outward direction.

Unilateral Bronchiolar Spasm. A patient with chronic bronchitis and emphysema and frequently recurring attacks of bronchiolar spasms had an attack of asthma in which the costal margin of the left side moved toward the median line, but the right

costal margin moved outward. The patient was given adrenalin, as on many former occasions, and its administration was followed by prompt relief of the asthma; directly the bronchiolar spasm ceased there was symmetrical outward movement of both costal margins. Unilateral bronchiolar spasm is a very unusual clinical experience, but this man gave us several exhibitions of this phenomenon, and in each instance the attack was relieved by adrenalin just as readily as when he had bronchiolar spasm throughout the lung.

When we are dealing with inspiratory retraction of the costal margin of one side, due to intrathoracic disease, a complete diagnosis is, of course, not directly implied, but we can be sure that the diaphragm has gained control of the costal margin. There is, however, an exception to this rule, and that is synechia between the diaphragm and the thoracic wall. If the pleural sinus is obliterated and the diaphragm has an attachment directly on the thoracic wall, then the insertions at the costal margin will no longer play a rôle in the control of the costal margin movement. Under these circumstances there will, of course, be no descent of the diaphragm, although the under surface of the arch of the diaphragm will be greatly accentuated in an upward direction, but the effective portion of the diaphragm will be that portion which lies between the central tendon and the synechia with the thoracic wall. Therefore the effective portion of the diaphragm may be horizontal when the concavity of the under surface is greatly increased. Under these circumstances the hypochondrium and costal margin will be drawn toward the median line during inspiration, although the leaf of the diaphragm on the affected side will occupy a much higher position than normally.

This movement of the costal margin can easily be shown experimentally on the dog by suturing the leaf of the diaphragm to a rib in the axillary and anterior axillary lines. This new fixation of the diaphragm will cause the costal margin to move toward the median line during inspiration. When the sutures are released the costal margin will again resume its outward movement. With this simple procedure the exact conditions of obsolete pleurisy with synechia between the diaphragm and the chest wall are reproduced.

Disparity of Movement Between the Inner and Outer Portions of the Costal Margins. The curve of the diaphragm varies in its different portions. The muscular fibers of the diaphragm which take their origin from the central tendon, and which are inserted along the costal margin from the costal angle to the eighth costal cartilage, have a much less convexity than the fibers from the lateral and posterior portion of the diaphragm which are inserted on the costal margin from the eighth rib downward and outward. In studying movements of the costal margin it is essential not only to observe the direction of movement and symmetry of movement near the subcostal angle but also to observe the move-

ment of the costal margin below and external to the end of the eighth rib. Should the pericardial sac be enlarged or should there be globular enlargement of the heart the subcardial portion of the diaphragm will be depressed. The anterolateral portions of the diaphragm have much less curve than the lateroposterior portions, and for this reason it requires much less depression of that part of the diaphragm to give its respective fibers mastery of the costal margins where they are inserted. In pericarditis with effusion, when the fluid accumulates in sufficient quantities to be a factor in circulatory disturbances, the subcostal angle will be symmetrically narrowed during inspiration. The costal margins on both sides, from the angle to the eighth rib, will be drawn toward the median line, but below and external to the eighth rib the costal margins on both sides will move away from the median line.

We have had many opportunities to confirm this in patients with pericarditis with effusion and in cases of mitral stenosis and myocarditis. Tubercular pericarditis gives a particularly good opportunity to confirm this observation. These patients require repeated paracentesis of the pericardial sac. When there is a large accumulation the subcostal angle will narrow symmetrically during inspiration, and as fluid is withdrawn the angle will resume its normal widening; with reaccumulation of fluid a return to inspiratory narrowing is again observed. This phenomenon is of considerable service in estimating the significance of pericardial effusion. In pericarditis with effusion attended by inspiratory widening of the subcostal angle there is no indication for paracentesis for relief of pressure within the pericardial sac. There may be other reasons for paracentesis, such as establishing drainage, but we may be quite sure that relief of pressure is not demanded so long as there is inspiratory widening of the subcostal angle.

In children, whose diaphragms are flatter than in adults, a very moderate globular enlargement of the heart, as seen in mitral stenosis, is quite sufficient to cause inspiratory narrowing of the subcostal angle. It is not an uncommon experience to find in elderly persons, in combination with myocardial and vascular disease, a chronic bronchitis and emphysema. Under these circumstances one may be sometimes left very much in doubt in an interpretation of the significance of pulmonary disease and cardiac disease as sources of respiratory discomfort. Under these circumstances the percussion of the borders of the heart is attended with some difficulty, but if there is an inspiratory narrowing of the subcostal angle and the costal margins on both sides below and external to the eighth rib move in an outward direction we may be quite sure that the patient's air hunger is due more to cardiac disease than to pulmonary disease. If the pulmonary emphysema were sufficient to cause air hunger when the patient is at rest the costal margins in their entire extent would move toward the median line. This observation has been

confirmed in many instances by seeing the entire costal margin resume a movement in an outward direction after the employment of adequate doses of digitalis, although the physical signs of emphysema remained the same.

Thus far I have found only one exception to this rule, in a case of pericarditis with effusion, where there was synechia between the epicardium and pericardium over the anterolateral aspects of the heart, and there was a saeculated purulent pericarditis which was restricted to the posterior aspect of the heart alone. In this case the subcostal angle did not narrow during inspiration, although seemingly at autopsy there was a sufficient amount of fluid in the pericardial sac to cause it.

Asymmetry in the Movement of the Subcostal Angle when the Lower and Outer Portions of the Margins Move Laterally during Inspiration. The most common exhibition of asymmetrical movement of the subcostal angle is seen in enlargement of the left side of the heart when the right auricle is not enlarged and the right ventricle does not share equally with the left ventricle in its enlargement. Aortic valve stenosis and insufficiency may be attended by marked enlargement of the left ventricle while the right ventricle retains its normal size. Under these circumstances if the subcostal angle is observed it will be seen that the left border from the angle to the eighth rib will move less in an outward direction than the symmetrical portion on the right side, and the left side may be drawn toward the median line during inspiration as the symmetrical portion on the right side moves outward. Patients with left-sided lesions of the heart who have developed incompetency, with dilatation of the right ventricle and right auricle, will exhibit symmetrical narrowing of the subcostal angle during the period of incompetency, but after rest and digitalis it will be seen that the left margin will continue to move toward the median line but the right margin will move outward.

The only instances thus far that I have encountered in which the right ventricle and right auricle were greatly dilated without enlargement of the left heart were in soldiers at a casualty clearing station during the period of pulmonary edema from phosgene-poisoning. These patients developed acute dilatation of the right ventricle and right auricle without enlargement of the left heart, and consequently the costal margin of the right side from the angle to the eighth rib moved toward the median line during inspiration, whereas the left side retained the normal outward movement. After the administration of adequate doses of digitalin, that is, $\frac{1}{75}$ grain hypodermically, the right heart regained its normal size and the subcostal angle resumed its normal symmetrical inspiratory widening. This was, of course, in patients who had only moderate emphysema with pulmonary edema and did not have bronchiolar spasm.

Careful study of the relative movements of the upper and inner

portions of the two costal margins is a very great aid in estimating the relative size of the left and right sides of the heart, unless there should be an increase in the intra-abdominal pressure to prevent depression of the subcardial portion of the diaphragm.

During the past summer a patient was admitted to Lakeside Hospital suffering from acute septic endocarditis. There were no evidences of enlargement of either the left or right side of the heart, but the diagnosis of septic endocarditis without localization was made on account of the successful culture of pneumococci from the venous blood. In the absence of any evidences of localized infection elsewhere in the body the diagnosis of endocarditis was made by exclusion. At autopsy septic thrombi were found on the tricuspid valve and pulmonary valve and also thrombi on the wall of the pulmonary artery about one-fourth inch above the site of the valve. Although this was primarily an endocarditis involving the tricuspid and pulmonary valves the left ventricle and right ventricle retained their normal size.

Paralysis of the intercostal muscles will also cause asymmetry in the movement of the subcostal angle. A patient had severe syringomyelia, with kyphoscoliosis of the dorsal vertebræ, marked atrophy of the trapezii and all the scapulohumeral muscles, moderate atrophy of the muscles of the arms and pronounced atrophy of the muscles of the forearms and intrinsic muscles of both hands. The upper left thorax was stationary during inspiration. There was no inspiratory excursion from the first to the seventh rib inclusive, but from the eighth to the twelfth ribs inclusive there was a normal inspiratory excursion. From the subcostal angle to the eighth costal cartilage of the left side the border moved toward the median line during inspiration. From the eighth rib downward the costal margin moved outward during inspiration. This asymmetry of movement was interpreted as due to paresis of the upper intercostal muscles of the left side. It was quite conceivable in a patient who had an extensive syringomyelia of the cervicodorsal cord.

Another patient, a young woman, aged twenty years, at fourteen years of age had had an attack of poliomyelitis which left her with permanent paralysis and atrophy of the forearms, intrinsic muscles of both hands and scapulohumeral muscles of both sides; also the thigh and leg muscles were extensively involved. On the right side from the second to the seventh rib inclusive there was marked retraction of the ribs during inspiration. The eighth rib was stationary, but from the ninth to the twelfth rib there was an inspiratory movement in a normal direction. The costal margin of the right side, from the angle to the eighth rib, moved toward the median line with inspiration; and although the ninth rib was firmly attached to the costal border, from the ninth to the twelfth the costal margin moved away from the median line during inspiration. This asymmetry of movement in the inner portions of the costal borders was inter-

preted as due to paralysis of the upper seven intercostal muscles of the right side. This lesion was apparently acquired at the time of her acute poliomyelitis.

The following case exhibited very unusual respiratory excursion of the costal margin. The patient had an old syphilitic disease of the myocardium and aorta, and in addition to this had acquired a tuberculous pleurisy of the left side. On entering the hospital it was found that during inspiration the right costal margin, from the angle to the eighth rib, moved toward the median line, and the left costal margin, from the angle to the eighth rib, moved very slightly away from the median line. The lower and outer part of the right margin moved away from the median line and the lower and outer portion of the left side moved toward the median line. After 1800 c.c. of bloody and turbid serum were removed from the left thorax, the entire right costal margin moved away from the median line during inspiration and so did the entire left costal margin, but it was observed that the upper and inner part of the left costal margin moved less in an outward direction than the margin of the right side. After the fluid had been removed a roentgen-ray picture revealed enlargement of the ascending and transverse arch of the aorta. The heart occupied a transverse position, but the base of the heart did not come to the right of the sternum. The diaphragm of the left side revealed a silhouette much higher than that on the right side and there was marked thickening of the pleura over the entire left side. Our interpretation of the excursion of the costal margins when he entered the hospital was that the retraction of the upper and inner portions of the right costal margin was due to depression of the right anterolateral portion of the diaphragm on account of the great cardiac displacement to the right. Prior to the thoracic paracentesis the right border of the heart came nearly to the right nipple line, and after paracentesis, when the heart came back to the normal position, the entire right border resumed its normal inspiratory excursion. On the left side the lower and outer portion of the costal margin resumed a normal outward movement during inspiration after the fluid had been withdrawn from the chest, but the upper and inner portion of the left costal margin retained about the same inspiratory movement before and after paracentesis on account of synechia between the anterolateral portion of the diaphragm and the thoracic wall, and this synechia did not permit the anterolateral portion of the diaphragm to be depressed when the pleural effusion was present, nor did it rise after the fluid was aspirated. This rather bizarre movement of the costal margin is described because it illustrates how accurately the curve of the diaphragm in its different portions is reflected by the inspiratory movement of the costal margins where the affected parts of the diaphragm are inserted.

SUMMARY. A summary of the whole matter is that in interpreting the inspiratory movements of the costal margins one must study the

symmetry and asymmetry not only of the entire costal margins but of the inner and outer portions of each costal margin. Movements of the costal margins are modified with changes in the curve of the plane of the diaphragm, by paresis of either the diaphragm or the intercostal muscles and by synechia between the diaphragm and the thoracic wall. Such studies improve the accuracy with which one differentiates between infraphrenic and supraphrenic disease, and enable one also to estimate the conformation of the heart and the size of the pericardial sac and to differentiate between lesions which cause phrenic displacement and those which do not modify the plane of the diaphragm.

EARLY LESIONS IN THE GALL-BLADDER.[1]

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The study of early pathological conditions in the gall-bladder has been greatly facilitated by cholecystectomy, an operation which many surgeons have deemed advisable in preference to cholecystostomy. In their experience many patients in whom the organ had been drained returned to them with symptoms unrelieved. The desired relief in such cases seems to have been accomplished, at least in a much higher percentage, by the secondary complete removal of the organ. This experience with such cases following a secondary cholecystectomy has led, in the last five years, to the custom of primary cholecystectomy in preference to cholecystostomy in patients with a visible lesion and also in some patients in whom there is no visible gross pathology but a definite clinical picture pointing to this organ, plus enlargement of lymphatic glands along the ducts.

From January 1, 1913, to January 1, 1919, 4998 gall-bladders were removed at the Mayo Clinic. Of these, 4824 (96.5 per cent.) showed unquestioned gross pathological lesions (Table 1).

In this series of conditions it may be seen there were 157 with slight lesions and 17 grossly "normal," most of which showed definite changes in the villi on examination with a high-power dissecting microscope or in microscopic sections.

[1] Presented before the American Gastro-enterological Association, Atlantic City, June, 1919.